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ORIGINAL ARTICLE

Less-invasive MR indices of clinically evident esophageal variceal bleeding in biliary atresia patients

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KEYWORDS
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Background/Purpose: Esophageal variceal hemorrhaging is potentially life threatening for long-term survivors of biliary atresia. We evaluated the feasibility of less-invasive parameters for predicting the presence of clinically significant esophageal variceal bleeding in biliary atresia patients.
Methods: A total of 30 patients aged 108–5314 days (median = 285 days) with biliary atresia underwent a magnetic resonance examination with fast spin-echo T2-weighted imaging and spin-echo, T1-weighted images with fat saturation after use of a contrast medium on a 1.5-tesla scanner. The splenic length-platelet ratio was divided by the each patient's body height (m) to produce the corrected splenic length-platelet ratios. In addition, the splenic volume index-to-platelet count ratio was divided by the patient's body weight (kg) to produce a corrected ratio.
Results: The corrected splenic length-platelet ratio was more significantly increased in 21 patients with esophageal variceal bleeding (Group A) than in nine patients without variceal bleeding [(Group B) 0.98 ± 0.64 vs. 0.44 ± 0.18, $p < 0.01$]. The splenic volume index-to-platelet count ratio corrected by body weight was significantly larger in Group A (510.7 ± 536.2) than in Group B (148.1 ± 88.9, $p < 0.01$).

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Conclusion: Less-invasive indices, including the corrected splenic length platelet ratio and the splenic volume index-to-platelet count ratio, may be valuable predictors of esophageal variceal bleeding in patients with biliary atresia.

Introduction

Biliary atresia (BA) is a progressive fibro-obliterative cholangiopathy that affects the extra- and intrahepatic biliary tree, resulting in fibrous obliteration of the biliary tract and the subsequent development of biliary cirrhosis. The development of the Kasai portoenterostomy procedure improves the prognosis and prolongs the lives of children with BA. However, portal hypertension still develops, even after the Kasai operation.[1,2] More than 40% of the long-term survivors had cirrhosis and/or portal hypertension.[2] Variceal bleeding is a common and life-threatening complication of portal hypertension in BA patients, displaying the highest mortality rate.[1–5]

Because esophageal variceal hemorrhaging can be devastating, the early diagnosis and follow-up of esophageal varices are mandatory in the postoperative care of patients with BA.[6,7] Several studies suggest less-invasive parameters for predicting the presence of esophageal varices in adults with liver cirrhosis,[8–28] but their validity in children with biliary cirrhosis is uncertain and has rarely been assessed. There have been few reports about less-invasive parameters for predicting esophageal variceal bleeding in patients with BA.[7] Therefore, this study was conducted with an aim of evaluating the feasibility of less-invasive magnetic resonance imaging (MRI) indices as indicators of the presence of esophageal variceal bleeding.

Patients and methods

Patient population

In this retrospective study, 31 patents with pathologically proven BA diagnoses and without cardiac anomalies received MR examinations from January 2006 to June 2010. One of the 31 patients had moderate ascites was excluded to avoid overestimating body weight. The 30 patients, consisted of 11 boys and 19 girls, underwent Kasai portoenterostomy between the ages of 26 and 147 (62 ± 23) days. MR examinations were performed at the age of 108–5314 days (median, 285 days). The 30 patients received abdominal MR examinations 33–5137 (median, 195) days after the Kasai operation, including 13/30 patients to look for possible complications of biliary cirrhosis and 17/30 patients for anatomic evaluation before liver transplantation. A patient received the Kasai operation at the age of 147 days and underwent an MR examination 33 days later due to persistent jaundice and a suspicion of portal hypertension. The summarized clinical demographic data of the 30 patients are listed on Table 1. This study was conducted according to a protocol approved by the Committee of Research Ethics of the National Taiwan University Hospital (201010044R).

Criteria for esophageal variceal bleeding

The 30 patients were subdivided into Groups A and B. The Group A patients had clinical signs of gastrointestinal bleeding and F1–F3 esophageal varices detected by panendoscopic examinations. Those patients without signs of gastrointestinal bleeding or esophageal varices on panendoscopy belonged to group B. A standard flexible gastrofiberscope (XP240 or XP260, Olympus, Tokyo, Japan) was used for a panendoscopic examination, no more than 4 months either before the MR examination, or after it. A total of 23 patients with clinical findings related to gastrointestinal bleeding (including fecal occult blood) received panendoscopic examinations. In two of the 23 patients with fecal occult blood, panendoscopic revealed no evident esophageal varices. A total of 21 patients had F1–F3 esophageal varices, according to Beppu classification,[29] confirmed by panendoscopic findings and were included in patient Group A. Among the 21 patients, seven patients had red color signs on the observed esophageal varices and the other 14 patients received variceal treatment including alcohol sclerosing therapy ($n = 1$), ligation ($n = 7$), and banding ($n = 6$). The latter 14 patients had diminished fecal occult blood after variceal treatment. In Group A, nine of the 21 patients were younger than 1 year of age.

The other seven of the 30 patients were younger than 2 years of age and had negative results of examinations of stool occult blood for at least one time. Four of the seven patients underwent living-related transplantation 2–6 months after MR examinations. Three of the seven patients received examinations of fecal occult blood every 6 months after the MR examinations for 20–40 months. The nine patients, including two with negative panendoscopic findings

Table 1 Demographic and laboratory data, Child-Turcotte-Pugh score, pediatric end-stage liver disease or model of end-stage liver disease score and clinical findings.

Sex (Male/Female)	10/20
Age at Kasai operation, d	62 ± 23
Body weight at MRI (kg)	16.0 ± 14.9
Bilirubin (g/dl)	10.2 ± 6.7
Albumin (mg/dl)	3.67 ± 0.43
Prothrombin time (second)	13.1 ± 2.7
Platelet count (1000/mm^3)	218 ± 157
Child-Turcotte-Pugh score	8 ± 1 (5–10)
Child-Turcotte-Pugh class (A/B/C)	5/23/2
Pediatric end-stage liver disease or model of end-stage liver disease score	12 ± 8 (−5–28)
Esophageal varices (±)	9/21
Shifting dullness (±)	24/6

MRI = magnetic resonance imaging.

of the lower esophagus and seven without fecal occult blood, were included in Group B.

Child-Turcotte-Pugh, pediatric end-stage liver disease, and model of end-stage liver disease scores

All clinical and laboratory data, including sex, age, body weight (kg), body height (cm), bilirubin level (g/dl), albumin level (mg/dl), prothrombin time (seconds), international normalized ratio (INR), and platelet counts (/mm^3), were obtained within 1 month before or after the MR examination. The Child-Turcotte-Pugh (CTP) score[30] was calculated using five variables: ascites, encephalopathy, bilirubin, albumin, and prothrombin time. The presence of ascites was diagnosed according to physical findings of shifting dullness. Values of 1, 2, or 3 were assigned to each of these variables to represent increasing severity, and a score was calculated as the sum of the five variables for each patient. A CTP score less than 7 was considered to be Class A; from 7–9 was Class B, while any score greater than 9 was Class C. The pediatric end-stage liver disease (PELD) scores[31,32] were calculated according to the following formula detailed in the homepage of the United Network for Organ Sharing (UNOS), and they were then multiplied by ten and rounded to the nearest integer, PELD score = $0.480 \times \text{Log}_e$ (bilirubin mg/dL) + $1.857 \times \text{Log}_e$ (INR) − $0.687 \times \text{Log}_e$ (albumin g/dL) + 0.436, if the patient is less than 1 year of age [scores for patients listed for liver transplantation before the patient's first birthday continued to include the value assigned for age (< 1 year) until the patient reached the age of 24 months], and + 0.667 if the patient has growth failure (< −2 standard deviations).

Model of end-stage liver disease (MELD) scores[31] were calculated using the following formula:

$$\begin{aligned}\text{MELD score} = {} & 0.957 \times \text{Log}_e(\text{creatinine mg/dL}) + 0.378 \\ & \times \text{Log}_e(\text{bilirubin mg/dL}) + 1.120 \\ & \times \text{Log}_e(\text{INR}) + 0.6431\end{aligned}$$

The PELD and MELD scores were multiplied by ten and rounded to the nearest whole number.

According to the latest UNOS modifications, laboratory values below 1.0 were rounded to 1.0 to avoid negative scores, and the maximum serum creatinine considered in the MELD equation was 4.0 mg/dL.

MRI

MRI was performed with a 1.5-tesla scanner (Signa Magnetom; GE Medical Systems, Milwaukee, WI, USA) using a phased-array torso. Out of the 31 patients younger than 6 years of age, 23 took chloral hydrate (National Taiwan University Hospital) per os with a dosage of 0.5 mg/kg body weight 10–45 minutes before their MRI examination. Proton-weighted imaging included the following sequences: fast spin-echo 4000-5000/8-12 (repetition time millisecond/echo time millisecond) with an imaging matrix of 256 × 192 or 256 pixels, a flip angle of 120–150, an echo-train length of 20, and one acquisition. T2-weighted imaging included the following sequences: breathing-averaged fast spin echo [time to repeat (TR)/time to echo (TE) = 3000-7000/90-100 millisecond (ms)] and breath-hold fast spin-echo (TR/TE = 2000–4000/70–100 ms) with a flip angle of 60–90, an echo-train length of 20, and one acquisition. T1-weighted spin-echo, breath-averaged (TR/TE = 500-700/10–20 ms, flip angle 90) images were also obtained with fat saturation in the coronal plane after using a contrast medium. The imaging matrix was 256 × 192 or 256 pixels for T2- and proton-weighed images and 512 × 512 for T1-weighted images. The slice thickness of proton-weighted, T2-weighted and T1-weighted images was 5 mm with an intersection gap of 1.5 mm.

Image measurement

The liver was divided into the left and right lobar (left medial, left lateral) and caudate regions. The caudate region was demarcated by an imaginary line connecting the bifurcation of the main portal vein and the right lateral border of the inferior vena cava. The measurements of the length or long and short axes (cm) of the left lobar, left medial, lateral, caudate hepatic regions and spleen were obtained from axial MRI according to previously described methods.[28,30] On the coronal T1-weighted spin-echo images, the largest cephalocaudal height (cm) of the spleen, left lobar, left medial, lateral, and caudate regions of the liver were measured. All three-axial length measurements of the hepatic regions and spleen were typically made within 1 cm of the level of the left umbilical portal vein and splenic hilum (Fig. 1A and B), respectively. Volume indices were calculated according to Ito and colleagues[30] as a simple product of these three diameters (cm) for the spleen and each region of the liver; i.e., (long-axis length) × (short-axis width) × (cephalocaudal height). These indices did not provide the exact measurements of hepatic regional or splenic volumes but gave an estimate based on the sizes of the hepatic regions. However, splenic measurement was not performed in one Group A patient who had received a splenectomy 1 year before MRI.

Less-invasive indices

We chose one measurement and five calculated ratio indices, including the maximal length of spleen (cm), the splenic length (cm)/platelet count (1000/mm^3) ratio (SLPR), the splenic volume index/platelet count (1000/mm^3) ratio (SVIPR), the volume index of the caudate lobe/albumin level (mg/dl) ratio, and the volume index of the left hepatic lobe/albumin level (mg/dl) ratio. To take the effect of age-related body weight and height into consideration, the splenic length and SLPR were further divided by the body height of each patient's body weight (kg) to produce a corrected splenic length and SLPR. In addition, the SVIPR, the volume index of caudate lobe-albumin level ratio and the volume index of the left hepatic lobe-albumin level ratio were divided by each patient's body weight to produce the corresponding corrected ratio indices.

Statistical analysis

All of the data were analyzed using Stata 8.0 software (IBM, Bryan, Texas, USA) and a p value less than 0.05 was

Figure 1 A 1482-day-old boy with biliary atresia after receiving Kasai operation. His platelet count was 66,000/mm^3 and his body weight and body height was 13 kg and 0.94 m, respectively, 4 days before the magnetic resonance imaging (MRI) examination. (A) axial fast-spin echo T2-weighted image; (B) spin-echo T1-weighted image in the coronal plane. The splenic length is 110.7 mm and the splenic volume index is 862.995. The corrected splenic length-platelet count and splenic volume index-platelet count ratios are 1.78 and 1.0058, respectively; (C) varices with red color signs (arrows) and a submucosal varix (arrowheads) are disclosed by a panendoscopy within the lower thoracic esophagus at a depth of 20 cm from the incisor three months after MRI examination.

considered statistically significant. The analysis of the biochemical data, image measurements and ratio indices, as well as the analysis of ordinal data such as sex, age, and score results, was performed using a Mann-Whitney rank-sum test. Multiple regression using an enter method of logistic model to include splenic length, splenic, and hepatic indices of BA patients with and without esophageal varices was performed. Sensitivity and specificity, as well as the best cut-off values for age, image measurements and index ratios for detecting the presence of esophageal variceal bleeding, were calculated using receiver operating characteristic (ROC) curves.

Results

The demographic parameters, image measurements, and less-invasive indices were correlated with the presence of esophageal variceal bleeding and listed in Table 2. Group A patients with esophageal variceal bleeding (Fig. 1C) were significantly older than Group B (1774 $\pm$ 1767 days vs. 219 $\pm$ 99 days, $p < 0.01$). The body weight and body height of patients in Group A (20.0 $\pm$ 16.3 kg and 0.98 $\pm$ 0.41 m) were significantly heavier and higher than that those assigned to Group B (6.8 $\pm$ 1.6 kg and 0.64 $\pm$ 0.07, $p = 0.04$ and 0.04, respectively). These results are shown in Table 2. In addition, Group A patients (198 $\pm$ 159 1000/mm^3) had higher platelet counts than Group B (310 $\pm$ 145 1000/mm^3, $p < 0.01$). However, CTP, PELD, and MELD scores, and the age when Kasai operation was performed did not vary significantly between patients in Groups A or B. None of the 23 chloral hydrate-sedated patients experienced respiratory distress or involuntary movement during MRI or within 30 minutes after it.

The length of the spleen in Group A patients (100.2 $\pm$ 34.2 cm) was significantly longer than in group B patients (72.4 $\pm$ 12.6 cm; $p = 0.02$). These results are also shown in Table 2. The SLPR was significantly larger in Group A than in Group B (1.071 $\pm$ 1.153 vs. 0.298 + 0.125, $p < 0.01$). After being corrected by the body height, the corrected SLPR was still significantly larger in Group A than in Group B (0.98 $\pm$ 0.64 vs. 0.44 $\pm$ 0.18, $p < 0.01$).

Before correction by the body weight, the SVIPR was significantly larger in Group A patients (14171 $\pm$ 25694) than Group B patients (1226 $\pm$ 839, $p < 0.01$). The ratios of the volume indices of both the caudate segment and the left hepatic lobe over the albumin level were also significantly larger (Fig. 1A and B) in Group A (19835 $\pm$ 28980 and 110672 $\pm$ 83310, respectively) than Group B (4529 $\pm$ 3461 and 55598 $\pm$ 25283, $p = 0.04$ and 0.02, respectively). After

Table 2 Comparison of demographic, laboratory data, Child-Turcotte-Pugh, pediatric end-stage liver disease or model of end-stage liver disease scores, splenic length, splenic and hepatic indices of biliary atresia patients with and without esophageal varices.

	Esophageal varices		*p* value
	(+)	(−)	
Sex			
Male	7	4	0.69
Female	14	6	
Age, d	1774 ± 1767	219 ± 99	<0.01*
Age when Kasai operation performed, d	66 ± 26	54 ± 9	0.49
Bilirubin (g/dl)	9.8 ± 7.7	10.9 ± 3.4	0.53
Body weight (kg)	20.0 ± 16.3	6.8 ± 1.6	0.04*
Body height (m)	0.98 ± 0.41	0.64 ± 0.07	0.04*
Albumin (mg/dl)	3.58 ± 0.44	3.86 ± 0.39	0.07
Prothrombin time (second)	13.6 ± 3.0	11.8 ± 1.4	0.13
Platelet count (1000/mm^3)	198 ± 159	310 ± 145	<0.01
Child-Turcotte-Pugh score	8 ± 2	7 ± 1	0.62
Pediatric end-stage liver disease or model of end-stage liver disease score	11 ± 9	12 ± 5	0.96
Length of spleen (mm)	100.2 ± 34.2	72.4 ± 12.6	0.02*
Length of spleen (mm)/body height (m)	11.23 ± 2.26	11.16 ± 2.81	0.56
Splenic length (mm)/platelet count (K)	1.071 ± 1.153	0.298 ± 0.125	<0.01*
Splenic length/body height (m)/platelet count	0.98 ± 0.64	0.44 ± 0.18	<0.01*
Splenic volume index (mm^3)/platelet count	14,171 ± 25,694	1226 ± 839	<0.01*
Splenic volume index/body weight (kg)/platelet count	510.7 ± 536.2	148.1 ± 88.9	<0.01*
Volume index of caudate segment/albumin level	19,835 ± 28,980	4529 ± 3461	0.04*
Volume index of caudate segment/albumin level/body weight	918 ± 750	701 + 554	0.50
Volume index of left hepatic lobe/albumin level	110,672 ± 83,310	55,598 ± 25,283	0.02*
Volume index of left hepatic lobe/albumin level/body weight	7152 ± 4141	9636 ± 4462	0.16

* = a *p*-value <0.05, indicating a statistically significant difference.

correction by the body weight, however, only the corrected SVIPR was significantly larger in Group A (510.7 ± 536.2) than Group B (148.1 ± 88.9, $p < 0.01$).

When the factors including age, body weight, length of spleen, SLPR, corrected SLPR, SVIPR, corrected SVIPR, and volume indices of both the caudate segment and the left hepatic lobe over albumin level were analyzed by enter method of logistic model (Table 3), only the corrected SLPR significantly predict the odds ratio of BA patients without over those with esophageal variceal bleeding ($p = 0.043$).

Table 3 Summary of splenic length, splenic and hepatic indices of biliary atresia patients with and without esophageal varices using the enter method of the logistic model.

Factor	Coefficient	Standard error	Wald value	Degree of freedom	*p* value	Odds ratio	Correlation strength
Age	−0.018	0.045	0.159	1	0.690	0.982	
Body weight (kg)	−0.405	0.974	0.173	1	0.678	1.989	Cox and Snell $R^2 = 0.658$
Platelet count (1000/mm^3)	−0.002	0.024	0.005	1	0.941	0.998	Nagelkerke $R^2 = 0.927$
Length of spleen (mm)	1.413	1.699	0.692	1	0.405	4.109	
Splenic length/platelet count	−207.031	359.963	0.331	1	0.565	0.000	
Splenic length/body height (m)/platelet count	−182.929	90.524	4.084	1	0.043	0.000	
Splenic volume index/platelet count	0.921	1.136	0.658	1	0.417	2.513	
Splenic volume index/body weight/platelet count	0.034	0.043	0.630	1	0.428	1.035	
Volume index of caudate segment/albumin level	−0.129	0.136	0.887	1	0.346	0.879	
Volume index of left hepatic lobe/albumin level	0.010	0.043	0.057	1	0.811	1.010	
Constant	1.479	10.368	0.020	1	0.887	4.387	
Global Model chi-square score	$X^2 = 31.146$; $p = 0.001$						
Goodness of fit	X^2 of Hosmer-Lemeshow test = 1.874, $p = 0.985$						

The ROC curve analysis of the corrected SLPR (area under curve = 1) showed significant discrimination between the patients in Groups A and B. A threshold of 0.506 was suggested for the corrected SLPR to achieve a specificity of 89% and a sensitivity of 100%. The area under curve of age was 0.67 and cut-off value of the age to be 195 days to reach the sensitivity of 85%.

Discussion

Even after a successful Kasai portoenterostomy, biliary cirrhosis can still progress in BA patients and induce esophageal varices. About two-thirds of BA children will have endoscopically visible esophageal varices by the time they are 2−3 years of age, and half of these will eventually bleed to death.[1,3−5] Because long-term survivors of BA are increasing, prompt and adequate management of esophageal varices is becoming increasingly important for the follow-up of postoperative BA patients to avoid serious and potentially devastating bleeding complications.[4,33]

A panendoscopy is currently the mainstay for diagnosing esophageal varices. However, there is no consensus on the timing for screening BA patients without signs related to esophageal varices.[7] In addition, a panendoscopy carries higher risk and entails more medical burden when performed on children than on adults because of the invasiveness of the procedure, anesthesia-related risks, and hospitalization costs. The treatment of esophageal variceal bleeding in children usually begins only after the first episode of gastrointestinal bleeding.[33] Our present protocol suggests that a panendoscopy be performed in patients without signs of gastrointestinal bleeding or portal hypertension after 2 years of age. However, our results suggested the cut-off value of the age to be 195 days to reach the sensitivity of 85%. A panendoscopy could be performed as early as 195 days to find out the asymptomatic esophageal varices earlier than before.

In some studies, PELD and MELD scores have been proven useful for assessing the severity of pediatric liver diseases, including that of BA.[34] However, neither the CPT nor the PELD/MELD score was significantly associated with the presence of esophageal variceal bleeding in our series. As a result, the severity of biliary cirrhosis as assessed by these two scores was not satisfactory in distinguishing Group A from Group B.[14,34]

A less-invasive procedure for identifying esophageal variceal bleeding will assist in determining the risk of variceal bleeding, choosing the optimal time to perform an endoscopy and avoiding potentially life-threatening variceal bleeding in children. Abdominal ultrasonography is the frequently used first-line modality to follow up the hepatic or splenic lesions in BA patients. However, it could be difficult to measure the exact three-dimensional diameters on a single ultrasonographic image when the spleen is huge. Most of the previously reported MR or computed tomographic measurements and parameters related to esophageal varices derive from studies of hepatitis-related cirrhotic adult patients.[8−28,30] However, adults and children are different in many ways and direct extrapolation of data related to esophageal variceal hemorrhages from adults to children may be unreliable.[33]

Because BA is a progressive disease, increasing age leads to higher incidence of esophageal variceal bleeding. However, age *per se* was not the only determinant of the development of esophageal variceal bleeding. The age when BA patients under Kasai operation were not significantly different between group A and B patients but the study did not include all the BA patients receiving Kasai operation during the study period. Similar to previous studies of cirrhotic adults, the ratio of the length of the spleen or the splenic volume index to the platelet count had high sensitivity for predicting the presence of esophageal variceal bleeding in BA.[8,9,11−13] Decreases in platelet count and increases in splenic length could be attributed to aggravated hypersplenism caused by progressing portal hypertension. In addition, the ratios of the volume indices of the caudate segment and left lobe to the albumin level were also significantly larger in patients with esophageal variceal bleeding.[35] The differences in the volume index-related ratios could partially relate to the differences in body weight between the Group A and Group B patients in our series. Since previous studies proposed that visceral organ weights increase with the total body weight in human neonates to adults,[36] we divided the SLPR and SVIPR by the body height and body weight, respectively, to obtain corrected ratios with sensitivity 100% for corrected SLPR. The SLPR per body height may be a good screening criterion irrespective of patient's age. Our two patients received an MR examination 1 and 3 months before the first attack of esophageal variceal bleeding. As a result, the less-invasive MR indices may be promising in predicting the presence of esophageal varices before a catastrophic hemorrhagic event.

Our study, however, has several limitations, including a small sample size and the possible overestimation of the exact incidence of esophageal variceal bleeding, because of possible concomitant bleeding from other gastrointestinal lesions. The indications and time-point of MRI examinations relative to panendoscopy and Kasai operation were quite heterogeneous in this retrospective study. Despite a good correlation between the corrected SLPR and the presence of esophageal variceal bleeding, it is difficult to rely on these indices alone to exclude patients from an initial screening endoscopy, especially patients with a remarkable weight gain due to a large amount of ascites. However, they may help to identify patients who could benefit from more frequent endoscopic follow-ups, especially in asymptomatic patients older than 195 days. The prospective use of less-invasive MR indices for screening of a well-selected group of BA patients could further validate the less-invasive indices. Although MRI examinations are expensive and sedation is usually necessary for uncooperative patients, measurement of corrected SLPR in BA patients with biliary or vascular complications requires no additional use of contrast medium and carries no increased risks but gives some clinical implication to further prophylactic measures.

In conclusion, indices including the SLPR per body height may be good predictors of esophageal variceal bleeding in patients with BA. We suggest that these easily accessible, less-invasive indices be used to help to identify BA patients who could benefit from earlier endoscopic follow-ups.

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